Diagnosis requires recording the degree and pattern of cognitive impairment, and for this there is no substitute for the neuropsychologic test battery administered by a psychologist aware of the possibility. The standard clinical neurologic examination usually gives normal results, except for those responses that suggest impaired mentation. Electroencephalograms are usually normal. Advanced cases often show reduced cerebral size on a computed tomographic scan.

The prognosis is grim, especially if the offending exposures have happened over a considerable time period. Rehabilitative efforts can aid in coping with the problems of living. Investigators in Sweden have found that solvent-induced encephalopathy has occurred even though the solvent exposures were within the recommended limits. Because of the similarity of the clinical pictures, cases of presenile dementia should be screened with a careful occupational history in a search for chronic solvent encephalopathy.

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REFERENCES

Baker EL, Letz R, Fidler A: A computer-administered neurobehavioral evaluation system for occupational and environmental epidemiology. J Occup Med 1985 Mar; 27:206-212

Elofsson SA, Gamberale F, Hindmarsh T, et al: Exposure to organic solvents—A cross-sectional epidemiologic investigation on occupationally exposed car and industrial spray painters with special reference to the nervous system. Scand J Work Environ Health 1980 Sep; 6:239-273

Fischbein A: Times change and diseases are changed in them. Am J Ind Med 1984; 5:261-263

Cataracts, Ultraviolet Light and Occupational Exposure

OVER THE PAST TEN YEARS, there has been a growing recognition among ophthalmologic scientists that a major factor in the progression of the cataract of advancing age is ultraviolet radiation exposure.

The origins of this concept are based in epidemiologic investigations. In the 19th and early 20th centuries, Gross and Schulek first observed that cataracts were more common among people working in fields than in cities. This was confirmed by ophthalmologists in Hungary, Germany, Romania and Egypt. Furthermore, in dwellers near the equator (India, Pakistan, Africa and Central and South America) cataracts occurred earlier and at greater frequency when compared with those in the temperate zones of Europe and North America. Current studies add to the evidence. In the 1970s it was reported that cataracts occur earlier and more often in persons in Israel and India than in those in England or Scotland. In the United States, Hiller and colleagues reported that cataract prevalence is correlated with the number of hours of daylight determined from weather maps. Zigman and associates studied three geographical areas and showed that where the sunlight is brightest, brown cataracts occur at a higher frequency. In workers exposed to sunlight (farming, fishing, truck driving and construction) nuclear brunescent cataracts were found to develop much more frequently than in those who worked indoors. An analysis of 100,000 Australians (Aborigines and whites) recently showed a clear correlation between the prevalence of senile brunescent cataracts and levels of ultraviolet radiation.

Long-wave or near-ultraviolet light (300 nm to 400 nm) is now known to damage the ocular lens by inducing molecular changes in its proteins and by causing cytopathologic changes in its constantly actively growing epithelial cells. Laboratory studies have elucidated the molecular basis of sunlight-induced cataracts. Photon absorption by aromatic amino acids in lens proteins, particularly tryptophan, has been shown to be an initial event. Abnormal pigments with decomposed tryptophan accumulate as a brown, nuclear cataract. On the other hand, cortical cataracts may have a different pathogenesis, involving photolysis of membrane transport enzymes.

The inclusion of an ultraviolet-blocking molecule in the lenses of safety or operating-driving glasses would be effective prevention. There are 111 million American workers in occupations with cataract-inducing levels of ultraviolet exposure.

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REFERENCES

Lerman S: Human ultraviolet radiation cataracts. Ophthalmic Res 1980; 12:303-314

Hiller R, Giacometti L, Yuen K: Sunlight and cataract: An epidemiologic investigation. Am J Epidemiol 1977; 105:450-459

Taylor HR: The environment and the lens. Br J Ophthalmol 1980 May; 64:303-310

Zigman S, Datiles M, Torczynski E: Sunlight and human cataracts. Invest Ophthalmol Vis Sci 1979 May; 18:462-467

Melanoma—An Occupational Hazard?

THE REPORTED INCIDENCE of skin melanoma is increasing among fair-complexioned people throughout the world. The increase is seen primarily between latitudes 45° north and 45° south. Higher levels of education and socioeconomic status, an indoor occupation, skin that sunburns easily, a history of melanoma or other skin cancer, dysplastic nevi and an increased number or size of moles are factors that correlate with melanoma incidence and in this sense may be thought of as "risk" factors.

Occupational factors have also been suggested as being contributory. However, when these suggested factors are examined among employee groups with comparable occupational exposure but in different geographic locations, consistency is lacking. At the First International Conference on Skin Melanoma, held in Venice in May 1985, neither the presentation of "Environmental Factors in Melanoma" by J. A. H. Lee, nor the subsequent discussions among the world experts in attendance implicated occupational factors as causal.

This leaves those concerned with melanoma the challenge of identifying factors possibly contributing to the increased frequency of melanoma diagnosis and of documenting possible causal relationships. Every practitioner of medicine must be vigilant in scrutinizing the skin of high-risk patients and seizing opportunities for the earliest possible detection of melanoma. Complete removal of thin lesions is almost always curative. Patient education is the cornerstone of early detection and consequent saving of lives.

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REFERENCES

Acquavella JF, Tietjen GL, Wilkinson GS, et al: Malignant melanoma incidence at the Los Alamos National Laboratory. Lancet 1982 Apr 17; 1:883-884

Austin DF, Reynolds P: A Case-Control Study of Malignant Melanoma Among Lawrence Livermore National Laboratory Employees. Berkeley, Calif, California Department of Health Services, report #3 (unpublished), Resource for Cancer, 1984 Jul 3

Friedman RJ, Rigel DS, Kopf AW: Early detection of malignant melanoma: The role of physician examination and self-examination of the skin. CA 1985 May-Jun; 35:130-151

Greene MH, Clark WH, Tucker MA, et al: Acquired precursors of cutaneous malignant melanoma: The familial dysplastic nevus syndrome. N Engl J Med 1985 Jan 10; 312:91-97

Wright WE, Peters JM, Mack TM: Organic chemicals and malignant melanoma. Am J IndMed 1983; 4:577-581

The Occupational History

MANY ARTICLES on occupational medicine remind physicians in other fields of the necessity of taking a good occupational history along with the medical history. Towards this end, various formats and styles of history taking are presented in the literature. Most tend to be highly inclusive, complex and cumbersome to use in daily practice. At the other extreme, a physican who does not use any routine or format may be lost early on and abandon the quest for occupational illness after the single inquiry, "What kind of work do you do?" And, finally, unless familiar with the field and legal requirements, many will not know what to do with the information gathered by using most available formats. The following format is presented to aid physicians both in taking a history and in directing them in the use of the information. The form should be used following a work history and positive response to general inquiry of exposure to substances and workplace environments such as asbestos, heavy metals, carcinogens, pesticides, noise, solvents, arsenic, ethylene oxide, dusts and radiation.

The format provides space for a general work description, exposure data and legal limits of toxic substances and required health surveillance in California. This format can be tailored to a particular area or population by adding or deleting specific exposure categories. It is best used when provided to patients in advance of their appointment so that reference can be made to work records for accuracy. However, it can also be used effectively when completed in the waiting room and reviewed with the examining physician at the same visit. It is most effective if maintained and compared from year to year on subsequent visits. The form is entitled "Interim Toxic

Cal/OSHA INTERIM TOXIC EXPOSURE HISTORY FORM TODAYS DATE:
WITH REFERENCE TO THE PAST THREE (3) YEARS:

NAME BIRTHDATE SOCIAL SECURITY #

OCCUPATION JOB TITLE JOB DESCRIPTION HOW LONG HELD

WORK RELATED INJURY OR INDUSTRIAL ACCIDENT?
PLEASE DESCRIBE:

EXPOSURE DATA: PLEASE BE ACCURATE

| FOR THE EXAMINING ** DOCTOR

SUBSTANCE/CODE	EXPOSURES DATE, DURATION, AMT	PROTECTIVE EQUIP WORN: YES	PROTECTIVE EQUIP WORN: NO	ACUTE EFFECTS: YES	ACUTE EFFECTS: NO	LEGAL LMT: 8 HR TWA/ ceiling	In addition to a work & medical Hx and P.E., ADD:
ASBESTOS/5208j						'action' 0.1 fiber /cc air PEL .5 fibers/cc	over 40:pa cxr, stool blood test, PFTs qlyr/ 3 view cxr q3yr under 40:pa cxr, PFTs q 1 year.
13 CARCINOGENS* 5209g						NO DIRECT CONTACT	ANNUAL EXAM
VINYL CHLORIDE 3201k						PPM/5PPM Ø ON SKIN	LIVER FUNCTION TESTS
DBCP/ 5212m						PPB/ Ø SKIN/EYE CONTACT	SERUM ESTROGEN/ SPERM CT; LH/FSH REPRODUCTIVE HX
ACRYLONITRILE/ 5213n						1 PPM	STOOL BLOOD, CXY DETAILED NEURO EX
MOCA/ 5215k 4'4 METHYLENEBIS (2-CHLOROANILINE)						10ug/cc3/ 50 """air surface: 100ug/cm	CXR PA,U/A,CBC, LIVER FUNCTIONS. MOCA URINARY TEST IF EXPOSED MORE THAN 10 YEARS.
EDB/ 5219k ETHYLENE DIBROMIDE						130PPB Ø LIQUID on eye/skin	UNDEFINED AT THIS TIME
LEAD 5215						50 ug/M3 "action" at 30ug/M	ZPP,BLOOD LEAD, CREATININE,CBC, BUN,U/A.
PESTICIDES/ ORGANOPHOSPHATES.						Individual limits	RBC and plasma cholinesterase.
NOISE						90dBA/115	ANNUAL AUDIOGRAM 'action level' at 85dBA
RESPIRATOR USE: TYPE: DATE:		WHY WORN:		FOR HOW LONG:			SPIROGRAM,EKG, HEARING/VISION
ARSENIC 5214						'action' level 5ug PEL 10ug/m3	CBC,U/A,CXR PA, SPUTUM CYTOLOGY
EtO 5220 ETHYLENE OXIDE						'action' Ø.5ppm PEL 1 ppm	CBC, REPRODUCTIVE HISTORY

*13 CARCINOGENS: Acetylaminofluorene,Aminodiphenyl,Benzidene,Dichlorobenzidene, Dimethylaminoazobenzene,alpha-Naphthylamine,beta-Naphthylamine,Nitrobiphenyl, Nitrosodimethylamine,beta-Propiolactone,bis-Chloromethyl ether,Methyl chloromethyl ether, Ethyleneimine.

**For further information regarding periodicity of examination and details of followup of abnormal data, consult the individual Sections cited.

Figure 1.—California Occupational Safety and Health Administration interim toxic exposure history form.